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Bariatric Surgery in a Case of Hypopituitary Patient with Craniopharyngioma: Management of Hypopituitarism and Long-Term Efficacy

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ABSTRACT

Craniopharyngiomas (CP) and their treatment are associated with weight gain often resulting in a syndrome of Hypothalamic Obesity (HO) resistant to dietary and medical interventions.

We report a case of successful bariatric surgery in a 21 years old female with panhypopituitarism and HO following CP treatment. Our patient obtained a weight loss (22 kg, -16.8%) at 6 months after Laparoscopic Sleeve Gastrectomy (LSG), a further reduction up to -36% after 11 yr, with weight loss maintained at the last follow-up (12 yr). LSG was associated with severe endocrine complications for a few months; hormonal replacement therapy and nutrient supplementation in the long term were required. Her metabolic profile and quality of life improved. We reviewed data from literature, confirming the efficacy of bariatric surgery in CP patients with similar weight loss after SG and Gastric Bay pass (GB), with serious adverse events only after GB. Hormonal therapeutic adjustments were required.

Bariatric surgery should be considered as a treatment option for morbidly obese CP patients. A multidisciplinary team is necessary to avoid endocrine and systemic complications, not only in the immediate post-operative period but also in the long-term follow-up.

Keywords: Craniopharyngioma; Hypothalamic obesity; Hypopituitarism; Bariatric surgery; Case report

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INTRODUCTION

Craniopharyngiomas (CPs) are rare brain tumours associated with endocrine or neurological complications. Damage to the hypothalamus, either by tumour bulk, surgical intervention, or radiation therapy can result in a syndrome of Hypothalamic Obesity (HO), which is typically a morbid obesity, strictly related to hypothalamic damage extent. [1-5] Weight gain occurs despite adequate endocrine replacement of pituitary hormone deficiencies and is resistant to lifestyle interventions, therefore leading to severe obesity in up to 55% of CP patients. [1,3,4] Obesity is associated with increased morbidity risk, it reduced quality of life (QoL), and increased mortality. [5] HO treatment is often difficult and the results discouraging. Dietary restriction and lifestyle modifications are often ineffective, as well as targeted pharmaceutical options. [4] Results of bariatric surgery are limited, but they offer promising results, although these patients experience greater risk, due to hormone deficiencies and neurologic disorders. [2,4,6,7] We report a case of a hypopituitary CP patient who underwent successful bariatric surgery. Moreover, we reviewed the literature to extrapolate indications on the management of hypopituitary patients with CP during bariatric surgery and its long-term follow-up.

CASE PRESENTATION

Our patient underwent Laparoscopic Sleeve Gastrectomy (LSG) at the age of 21 years for severe obesity (height 156 cm; weight 131.2 kg; body mass index (BMI) 53.9 kg/m2; waist circumference 111 cm; hip circumference 135 cm) with associated hyperinsulinism (HOMA-IR=5.4), hypercholesterolemia, hyperuricemia, fatty liver grade 1, flat foot, valgus knee, and psychological distress. She had a history of complete surgical removal (at the age of 9 years) of a suprasellar CP with intraventricular development, with consequent hypopituitarism, diabetes insipidus and severe obesity, which had been refractory to treatment with integrated rehabilitation protocol, metformin, and hospitalized diet-exercise program. Magnetic resonance imaging showed an encephalo-malacic cavity with Cerebro Spinal Fluid (CSF) content in the right side of hypothalamus, supporting the diagnosis of HO.

At time of LSG corticosteroids were administered with the standard approach to pan-hypopituitary patients who undergo major surgery. Desmopressin dose was increased, on the basis of clinical parameters. The patient received education concerning postoperative nutritional needs; however, after discharge from surgery, she required urgent hospitalization for severe dehydration and adrenal crisis. She then developed a Central Venous Catheter (CVC)-related Candida sepsis that needed intensive care. After adjustment of the therapeutic regimen, the patient was discharged, but nausea and food aversion persisted, requiring one-day hospitalization to provide parenteral hydration and nutrient supplementation. Three months after surgery she was readmitted to our hospital for dehydration and severe hypernatremia. At discharge, the patient showed normal fluid balance using intranasal desmopressin and a good clinical status with adequate hormone

replacement (she refused somatotropin) and micronutrient supplementation. Anemia reappeared, requiring intravenous martial therapy for two years. Normalization of haemoglobin was achieved only after 8 years and currently persists with vitamin complex B supplementation. Vitamin D levels have also been maintained normal with oral cholecalciferol. Two years after LSG the patient underwent laparoscopic cholecystectomy for lithiasis, without complications.

LSG was followed by a rapid weight loss of 22 kg at 6 months (-16.8%), with additional loss of 2 kg at 12 months (-18.3%). (Table 1) Subsequently, there was a slight gradual increase in weight up to 111.2 Kg at 36 months. This peak was followed by further progressive weight loss of 25 kg (due to calorie restriction with dietician monitoring, in combination with physical exercise) reaching a body weight of 95 kg (-27.6 %; BMI 39 Kg/m^2) at 8 years and of 84 kg (-36%; BMI 34.52 Kg/m^2) at 11 years after LSG. Then the weight remained stable up to the last follow-up at 12 yr

Table 1: Clinical and biochemical characteristics of the patient before and after bariatric surgery.

	Baseline	After LSG	Follow-up 3° month	Follow-up 6° month	Follow-up 12° month	Follow-up 18° month	Follow-up 24° month	Follow-up 3° year	Follow-up 5° year	Follow-up 8° year	Follow-up 12° year
Body weight (Kg) /	131.2 /	130 /	116 /	109 /	107 /	105 /	105.5 /	111.2 /	109 /	95 /	84 /
BMI (kg/m^2)	53.9	53.4	47.6	44.8	43.9	43.1	43.3	45.7	44.8	39.03	34.5
HOMA index	5.4				1.9	1.1	1.5	1.1	2.3	1	1.5
Glucose (mg/dl)	76	112	95	69	80	70	73	69	86	68	74
Insulin (mUI/l)	29.1				9.9	6.5	8.2	6.8	11	6.1	8.2
Uric acid (mg/dl)	8.28	12.1	6.5	6.5	6.6	6.4	7	6.6	6.9	6.3	6.8
Total cholesterol (mg/dl)	240	294	250	241	261	271	305	266	249	241	199
Triglycerides (mg/dl)	189	172	155	95	192	147	174	169	126	117	122
LDL- cholesterol (mg/dl)	231.2	199	151	151	135	158	184	143	145	136	115
HDL-cholesterol (mg/dl)	71	61	68	71	87	84	86	89	78	81	59
ALT (U/L)	19	243	45	16	18	24	21	34	64	18	23
Calcium (mmol/l)	2.41	1.99	1.92	2.18	2.31	2	2.34	2.35	2.21	1.99	2.28

Phosphorus (mg/dl)	4.3	2.1	3.5	4	3.2	3.4	3.1	3.2	2.52	3	3.4
Vitamin D (ng/ml)	15	14.3	21.2	28.4	24.7	36.3	25.9	26.4	34.2	30.7	31.7
Haemoglobin (g/dl)	12	8.9	11.6	9.5	9.3	9	12	10.3	11.1	12.7	13.1
Creatinine (mg/dl)	0.63	0.7	0.6	0.62	0.74	0.6	0.7	0.84	0.66	0.72	0.8
Sodium (mEq/l)	144	155	148	144	144	140	140	141	142	144	143
Potassium (mEq/l)	4.42	3.8	3	4	4.5	4.1	4.4	4.4	3.5	3.8	4.2
Serum osmolarity (mOsm/Kg)	297	302	319	285	288	283	285	275	290	300	334
Urine osmolarity (mOsm/Kg)	421	283	95	583	658	659	505	605	498	580	652
FT4 (ng/dl)	1.29	0.83	0.76	1.42	1.64	1.14	1.36	1.21	1.6	1.33	1.38
IGF-1 (ng/ml)	41.4	33.2	44.5	42.3	35.1	28.6	34.3	31.5	30.2	33.4	28

Weight loss was associated with normalization of HOMA index and uricemia. Wellbeing, hunger control, satiety, and psychological condition improved, and the patient advanced her education by attending technology courses, consequently obtaining a high school diploma and a job.

Hormonal replacement therapy was modified with time (Table 2): hydrocortisone was maintained at the daily dose of 40 mg during the first months, then it was reduced to 30 mg at 6 months and to 20 mg at 3 years. The daily dose was maintained until the 12th year, with temporary dose increases to 25 mg/day according to the clinical condition. L-thyroxin (L-T4) dose was initially increased, and then decreased from the third month. In the first months after LSG the patient switched from sublingual to intranasal desmopressin formulation due to dehydration episodes. Then, the dose of intranasal desmopressin was reduced.

Table 2: Changes in oral hormonal supplementation after bariatric surgery.

Ethinylestradiol, EE; Estradiol Hemihydrate, EH; Dydrogesteron, DHG; Norelgestromin, NE; Transdermal, TD; Sublingual, SL; Intranasal, IN.

	Baseline	**After LSG**	**Follow-up 3° month**	**Follow-up 6° month**	**Follow-up 12° month**	**Follow-up 18° month**	**Follow-up 24° month**	**Follow-up 3° year**	**Follow-up 5° year**	**Follow-up 8° year**	**Follow-up 12° year**
Desmopressin	120 µg/die, SL	120 µg/die, SL	10 µg/die, IN	5 µg/die, IN	5 µg/die, IN	5 µg/die, IN	5 µg/die, IN	5 µg/die, IN	5 µg/die, IN	5 µg/die, IN	5 µg/die, IN
Hydrocortisone	20 mg/die	40 mg/die	40 mg/die	30 mg/die	30 mg/die	30 mg/die	30 mg/die	20 mg/die	20 mg/die	20 mg/die	20 mg/die
L-tiroxine	1050 µg/week	1050 µg/week	800 µg/week	800 µg/week	750 µg/week	750 µg/week	825 µg/week	825 µg/week	875 µg/week	750 µg/week	625 µg/week
Hormone replacement therapy (HRT)	EE 50 µg/die, TD for 21 days, TD	EE 50 µg/die, TD for 21 days, TD	EE 50 µg/die, TD for 21 days, TD	EE 50 µg/die, TD for 21 days, TD	EE 33.9 µg/die	EE 33.9 µg/die	EE 33.9 µg/die	EE 33.9 µg/die	EE 33.9 µg/die	EE 33.9 µg/die	EH 25 µg/die for 21 days, TD
	DHG 10 mg for 10 days/month	DHG 10 mg for 10 days/month	DHG 10 mg for 10 days/month	DHG 10 mg for 10 days/month	+ NE 203 µg/die, for 21 days, TD	+ NE 203 µg/die, for 21 days, TD	+ NE 203 µg/die, for 21 days, TD	+ NE 203 µg/die, for 21 days, TD	+ NE 203 µg/die, for 21 days, TD	+ NE 203 µg/die, for 21 days, TD	DHG 10 mg for 10 days/month

DISCUSSION

We showed that LSG may be effective in long term treatment of CP patients, but it may be associated with severe endocrine complications in the first months after surgery.

As suggested by the literature, [7] our patient received a standard approach to glucocorticoid treatment during bariatric surgery and hydrocortisone was maintained at higher doses at discharge as compared to those before surgery. However, she presented an adrenal crisis and severe dehydration immediately after discharge from surgery and she needed two readmissions during the following 3 months for severe dehydration. Hospitalization for suspected adrenal crisis was reported in another case after Laparoscopic Adjustable Gastric Band (LAGB), [8-17] suggesting special attention in these patients not only in the first few days after surgery but also in the first months.

Little is known about the effects of bariatric surgery on glucocorticoid absorption. In patients with primary or secondary adrenal insufficiency, plasma cortisol profiles after hydrocortisone were similar before and after bariatric surgery, moreover only minor differences were observed among different types of surgery, suggesting using a postoperative cortisol profiling to guide appropriate glucocorticoid dose adjustment. [18]

It has been shown that cortisol clearance is inversely related to insulin sensitivity and that fatty liver disease is associated with increased cortisol clearance. [19] Therefore, cortisol clearance and cortisol distribution can be expected to decrease after weight loss, requiring a lower glucocorticoid replacement dose. [21] However, no substantial changes in oral glucocorticoid dose were reported after bariatric surgery in CP patients. (Table 3)

In our patient, L-T4 dose was progressively reduced, in agreement with previous reports of CP patients who received either LSG or GB. [11,13] In contrast, other Authors showed no change or slight increase in L-T4 dose after GB. [11]

Table 3: Summary of data from literature describing a total of 40 patients receiving bariatric surgery for hypotalamic obesity after craniopahryngioma. Laparoscopic-adjustable gastric banding (LABG); laparoscopic sleeve gastrectomy (LSG); gastric bypass (GB); biliopancreatic diversion (BPD).

Authors	Number/(sex)	Bariatric procedure median age (range)	Bariatric procedures (number)	Adverse event	Endocrine complications	Hormonal replacement change	Follow-up time range and/or median	Responders, number BMI reduction or weight loss, median (range)

Gatta et al, 2013	4 (2F)	LSG 33.5 yr (24-43) GB	LSG (2)	Klebsiella septicemia*, esophagus ulcerations* (n = 1, GB)	Diabetes insipidus crisis * (n=1, GB)	None reported	2.5 yr LSG	2/2 SLG BMI reduction: 14.6% (19.6-9.6)
		40.7 yr (30.5-51)	GB (2)				4–5.3 yr median 4.7 GB	1/2 GB BMI reduction: 14.1%
Weissman et al. 2013	9 (7F)	17 yr (12-30)	LAGB (6)	Dysphagia and/or abdominal pain and/or vomiting (n=6 LAGB)	Hospitalization for suspected adrenal crisis* (n=1, LAGB)	Impaired effectiveness of oral desmopressin (n=1)	1-9 yr, median 5,5 LAGB	6/6 LAGB, 4/4 LSG No weight loss, but weight stabilization
			LSG (2 +2 12 months after LAGB)	Re-adjustement of binding (n=2 LAGB)		Hydrocortisone dose was transiently increased (n=2)	0,4-4 yr, median 2 LSG	2/2 GB weight loss: 30%
			GB (1 +1 12 months after LAGB)	Device was finally explanted (n=4 LAGB)			2-4 yr, median 3 yr GB	
Wolf et al., 2016	4 (3F)	20.5 yr (16-26)	GB	None reported	Marked reduction in fT4 concentra-tion in the long-term follow-up (n=1) Decreased IGF-1 levels	Therapy was slightly adapted following gastric bypass surgery compared to baseline (n=3)	1-5.4 yr, median 3.2	4/4 GB BMI reduction and weight loss: ~27 %
						Hydrocortisone		

						dose was distinctly reduced (n=1) Hydrocortisone dose was marginally increased (n=2) L-thyroxine dose was markedly reduced (n=1) L-thyroxine dose was slightly increased (n=1)		
Trotta et al., 2017	3 (2F)	22.3 yr (21–24)	LSG	Minor bleeding (n=1), mild folic acid and vitamin D deficiency (n= unknown)	None reported	L-thyroxine dose was reduced (n=2) Desmopressin dose was reduced (n=3) GH dose was increased (n=2) GH was Started (n=1)	2 yr	3/3 LSG BMI reduction: 28.25 %
Garrez et. Al, 2020	5 (4F)	LSG 34 yr (27-40) GB 38 yr (36-47)	LSG (3) GB (2)	Slight abdominal pain, vomiting and diarrhoea Epigastric pain, vomiting of greater	None reported	Minor adjustment in HRT (n=5)	2 yr	2/3 LSG 2/2 GB weight loss: 30%

				magnitude (n=1, GB)				
Wijnen et al., 2017	8 (7F)	13 yr (2-26)	LSG (3) GB (4+1 after 2 yr LSG)	Non reported	Non reported	Minor adjustments in hormone replacement (n=3)	2 yr	5/5 GB weight loss: 25%
						Minor increase recombinant human growth hormone dose (n=3)		
						L-thyroxine dose was reduced (n=3)		
						desmopressin dose was slightly increased (n=2)		2/3 LSG weight loss :10%
						Estradiol and progesteone dose was reduced (n=1)		
Inge et al., 2007	1 (M)	18 yr	GB with truncal anterior vagotomy	Mild iron-deficiency anemia	None reported	standard hormone replacement for hypopituitarism was continued	2.5 yr	BMI reduction was 21.6 %
Page-Wilson et al., 2012	1 (F)	18 yr	GB	None reported	None reported	None reported	1.6 yr	Weight loss: 26.6%
								BMI reduction:

								24.4 %
Rottembourg et al., 2009	2 (1F)	GB 12.7 yr	GB (1)	Diarrhea, chronic pain, fatigue, dumping-type syndrome*, and psychologic deterioration (n=1, GB)	None reported	not require changes in doses of medications	2-4 yr median 3 yr	GB BMI reduction: 33.8%
		BPD 15 yr	BPD (1)	Acute gallstone pancreatitis* (n=1, GB)		required pubertal induction		BPD BMI reduction: 28.8%
				Symptomatic hyperuricemia (n=1, GB)		GH was started		
				Very serious bradycardia necessitating pacemaker and intestinal stenosis with bleeding requiring laparoscopic surgical repairs* (n=1, BPD)				
				Recurrent fever				

				(n=1, BPD)				
Bretault et al., 2016	3 (1F)	29 yr (19-48)	GB (2)	None reported	None reported	None reported	1.5 yr	GB weight loss: 14%
			LSG (1)					LSG weight loss: 9 %

These data suggest that oral hormone replacement (hydrocortisone and L-T4) must be personalized and closely monitored not only in the first months after surgery but also in the long term. [21,22]

In CP patient's nausea, vomiting and/or lack of thirst may favour electrolytes imbalance and severe dehydration possibly leading to neurological complications. Dehydration may be a common complication of bariatric surgery in normal obese, requiring treatment especially in GB (4.8%) compared with SG (3.3%) patients. [23] As in our patient, presence of diabetes insipidus may favour and worsen this complication requiring readmission and increasing the risk of neurological complication due to severe hypernatremia. Our patient needed parenteral hydration for a long time, associated with change of desmopressin formulation. [11]

These data confirm that the appropriate medical management of post-bariatric follow-up requires particular attention in CP patients, to avoid endocrine complication.

In the general population, SG is associated with fewer nutrient deficiencies as compared with GB. [24] We observed iron-deficiency anemia requiring intravenous and oral therapy for two and five years, respectively, confirming the need of a long-lasting monitoring of micronutrient deficiencies in CP patients.

Despite the endocrine complications, LSG has been associated with a successful clinical outcome in our patient leading to a rapid weight loss of 22 kg (-16.8%) in the first 6 months, followed by progressive fall in body weight up to a 36% reduction. Data from literature confirmed that CP patients may have a good response to SG obtaining a median weight loss of 11.2% (range 9 to 14.7) and/or BMI reduction of 21.4% (range 14.6 to 28.2). (Table 3) The number of responders to SG appeared to be lower as compared with GB (7 out of 16 vs. 1 out of 20, p=0.012). However, the majority of non-responders obtained a stabilization of body weight after SG, which could be considered a positive result in these patients. Some authors suggested that non-adjustment of

hydrocortisone doses may play a role in the suboptimal weight loss in some CP patients after SG. [12] However, few patients with short follow-up time (mean of 2 years for SG and 2.7 years for GB; >5 years in a few cases) are reported in literature, hampering the possibility to draw definitive conclusions concerning long-term efficacy of SG and/or GB in CP patients.

It is known that SG promotes weight reduction via two main mechanisms: one is stomach restriction; the other is appetite reduction due to changes in the levels of gut hormones (ghrelin, GLP-1, insulin). [17,24] HO is characterized by increased parasympathetic activity with hyperinsulinemia, insulin resistance and subsequent increased visceral fat accumulation, which may result in severe obesity. [4] Other studies demonstrated a reduced postprandial suppression of ghrelin and/or a reduced postprandial response of PYY in CP patients, suggesting a role of these factors for disturbed regulation of appetite and severe obesity. [25,26] Therefore, the second mechanism seems to play an important role in contributing to weight reduction and long-term maintenance of weight loss in CP patients.

Data from literature suggest that SG may be associated with minor post-operative complications compared with GB in CP patients, (Table 3) as in the general population. [24] Moreover, no increased mortality was observed after both SG and GB in CP patients. [8-17] By contrast, our patient presented serious postoperative complications and an electrolyte imbalance which lasted for a few months, highlighting the importance of an adequate post-operative management to avoid severe complications in hypopituitary CP patients. Impaired absorption of oral hormone replacement therapy in these patients can increase the risk of mortality for acute adrenal insufficiency and/or electrolytes imbalance and severe hypothyroidism.

In conclusion, our report and the available literature data suggest that bariatric surgery may be a treatment option for morbidly obese CP patients, but they must be referred to a specialized bariatric surgical centre with a dedicated multidisciplinary team. Indeed, CP patients need hormonal replacement therapy adjustments that should be individualized according to clinical data and daily profiles, before, as well as after bariatric surgery and in the long-term.

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